UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/729,056	12/05/2003	David J. Grainger	295.009US4	9616

45837 7590 07/03/2007

SCHWEGMAN, LUNDBERG, WOESSNER & KLUTH/NEORX
PO BOX 2938
MINNEAPOLIS, MN 55402

EXAMINER	
RAMACHANDRAN, UMAMAHESWARI	
ART UNIT	PAPER NUMBER
1617	

MAIL DATE	DELIVERY MODE
07/03/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/729,056	GRAINGER ET AL.	
	Examiner	Art Unit	
	Umamaheswari Ramachandran	1617	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *21 May 2007*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *153-173* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *153-173* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Applicant's election of species, cardiovascular indication and idoxifene in the reply filed on 5/12/2007 is acknowledged. The restriction election has been made with traverse. The species election is withdrawn in consideration of Applicants' remarks and due to prior art related to the subject area of TGF-beta elevation and vascular indications. Claims 153-173 are pending.

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 153-173 are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 173-194,196-203, 205-211 and 231 of copending Application No. 09/754,775. Although the conflicting claims are not identical, they are not patentably distinct from each other because the copending application teaches an aspect of the claims in the instant application. For example, the method claimed in claims 153-173 of the instant application utilizes the same biological pathway comprising increasing the level of TGF-beta encompassing utilizes the same active agents in the method of claim 173 of the co-pending application.

The instant application teaches the method of treatment of vascular indication administering the therapeutic agents claimed by Applicants in the co-pending application and hence renders obvious over the diseases and the agents claimed in the co-pending application.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

Claims 153-173 are rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims of U.S. Patent Nos. 5,472,985, 5,770,609, 5,5595722, 5,559,844, 5,773,479, 5,847,007, 6,074,659, 6,166,090, 6,197,789, 6,262,079, 6,251,920. Although the conflicting claims are not identical, they are not patentably distinct from each other because the examined claims are either anticipated by, or would have been obvious over, the reference claims. Here claims 153-173 are generic to all that is recited in claims of cited U.S. Patents. That is, claims of cited U.S. Patents fall within the scope of claims 153-173. The instant application teach a therapeutic method of preventing or treating a vascular indication in a mammal which indication is characterized by a decreased lumen diameter, comprising: a) selecting an agent for TGF-beta elevation; b) administering a cytostatic dose of the agent to the mammal so as to inhibit smooth muscle cell proliferation, inhibit lipid accumulation, increase plaque stability, or any combination thereof. The cited patents teach methods of treating vascular indications such as atherosclerosis or inhibiting the pathological proliferation of mammalian smooth muscle cells, treating procedural vascular trauma, treating diabetics, administering compounds that elevate TGF-beta

factor. Specifically the method of treatment for vascular indications and by administration of same therapeutic agents or their analogs is claimed in both the instant application and cited U.S. Patents.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 153-168 are rejected under 35 U.S.C. 112, first paragraph, because the specification does not reasonably provide enablement for preventing a vascular indication in a mammal. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

The instant specification fails to provide information that would allow the skilled artisan to practice the instant invention without undue experimentation. Attention is directed to *In re Wands*, 8 USPQ2d 1400 (CAFC 1988) at 1404 where the court set forth the eight factors to consider when assessing if a disclosure would have required undue experimentation. Citing *Ex parte Forman*, 230 USPQ 546 (BdApls 1986) at 547 the court recited eight factors: (1) the nature of the invention; (2) the state of the prior art; (3) the relative skill of those in the ad; (4) the predictability or unpredictability of the art; (5) the breadth of the claims; (6) the amount of direction or guidance presented; (7) the presence or absence of working examples; and (8) the quantity of experimentation necessary.

(1) The Nature of the Invention:

The rejected claims are drawn to a therapeutic method for preventing or treating a vascular indication in a mammal which indication is characterized by a decreased lumen diameter, comprising: a) selecting an agent for TGF-beta elevation; b) administering a cytostatic dose of the agent to the mammal so as to inhibit smooth muscle cell proliferation, inhibit lipid accumulation, increase plaque stability, or any combination thereof.

(2) Breadth of the Claims:

The instant claims are broad and embrace preventing or treating a vascular indication in a mammal which indication is characterized by a decreased lumen diameter, comprising: a) selecting an agent for TGF-beta elevation; b) administering a cytostatic dose of the agent to the mammal so as to inhibit smooth muscle cell proliferation, inhibit lipid accumulation, increase plaque stability, or any combination thereof.

(3) Guidance of the Specification:

The guidance of the specification is towards the prevention of vascular indication administering TGF-beta agent is completely lacking.

(4) Working Examples:

Applicant does not provide any working examples for the prevention of vascular indication administering TGF-beta agent in a mammal.

(5) State/predictability of the Art:

The state of the art regarding treating a vascular indication administering TGF-beta agent in a mammal is relatively high. However, the state of the art for prevention of vascular indication administering TGF-beta agent in a mammal is underdeveloped.

(6) The Quantity of Experimentation Necessary:

The instant claims read on the prevention of vascular indication administering TGF-beta agent in a mammal. As discussed above, the specification fails to provide sufficient support for completely protecting a mammal against vascular indication. Applicant fails to provide information sufficient to practice the claimed invention, absent undue experimentation. Genetech, 108 F.3d at 1366 states that "a patent is not a hunting license. It is not a reward for search, but compensation for its successful conclusion" and "patent protection is granted in return for an enabling disclosure of an invention, not for vague intimations of general ideas that may or may not be workable."

Accordingly the claims are evaluated as a method for treating a vascular indication in a mammal and not as a method for preventing a vascular indication in a mammal.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 153-155, 157-173 are rejected under 35 U.S.C. 103(a) as being unpatentable over Grainger et al (Biochem J, 1993, 294, 109-112) in view of Nuovo et al. (Int J of Gynecological Pathology, p 125-131, 1989) and further in view of Purchio et al. (U.S. 5,221,620).

Grainger et al. teach the induction of Transforming Growth Factor (TGF) β levels and the decrease in the rate of proliferation of rat vascular smooth muscle cells in culture (see Abstract) by tamoxifen.

The reference does not teach a method of treating a vascular condition in a mammal characterized by a decreased lumen diameter.

Nuovo et al. teach administration of tamoxifen for treatment of endometrial polyps in postmenopausal patients with features including thick-walled blood vessels. (abstract). The patient (at risk of or afflicted with vascular indication characterized by a decreased lumen vessel diameter), condition (thick-walled blood vessels encompassing decreased lumen vessel diameter) to be treated and the effect are the same. An explanation of why that effect occurs does not make novel the same treatment of the conditions encompassed by the claims. The reference teaches a dose of 20 mg/day of tamoxifen (case 1).

The references do not teach that the agent in the treatment of vascular indication increases the production of TGF- β mRNA levels.

Purchio et al. teach that tamoxifen treated PC-3 cells contain more TGF-β mRNA.

It would have been obvious to one of ordinary skill in the art at the time of the invention to treat a vascular indication by the administration of an agent that elevates TGF-β levels. The motivation to do so is provided by Grainger et al. One of ordinary skill in the art would have been motivated to treat a vascular condition because the reference clearly teaches that an agent like tamoxifen decreased the rate of proliferation of rat vascular smooth muscle cells and induces TGF-β levels in culture. The references do not specifically teach determining an agent for TGF-beta elevation or selecting a cytostatic dose of the agent as claimed by applicant. The dosage or selection of an agent or mode of administration is clearly a result effective parameter that a person of ordinary skill in the art would routinely optimize. Optimization of parameters is a routine practice that would be obvious for a person of ordinary skill in the art to employ. It would have been customary for an artisan of ordinary skill to determine the optimal amount of ingredient to add in order to best achieve the desired results. The references do not teach that the smooth muscle cell proliferation is associated with procedural vascular trauma. It would have been obvious to one of ordinary skill in the art to administer an agent in a vascular indication that is associated with procedural vascular trauma. One of ordinary skill in the art would have been motivated to administer an agent that has been shown by Grainger that decreases the proliferation of smooth muscle cells in general to any condition related to smooth muscle cell proliferation in order to achieve similar therapeutic benefits.

Claims 153-155, 157-173 are rejected under 35 U.S.C. 103(a) as being unpatentable over Grainger et al (Biochem J, 1993, 294, 109-112) in view of Bjorkerud

(Arteriosclerosis and Thrombosis, 1991, 11, 892-902) and further in view of Purchio et al. (U.S. 5,221,620).

Grainger et al. teach the induction of Transforming Growth Factor β levels and the decrease in the rate of proliferation of rat vascular smooth muscle cells in culture (see Abstract) by tamoxifen.

The reference does not teach a method of treating a vascular condition in a mammal characterized by a decreased lumen diameter.

Bjorkerud teach the effects of TGF- β on human arterial smooth muscle cells. The reference teach that TGF- β promotes the myodifferentiation and inhibition of growth of smooth muscle cells in vitro and could play an important role in the arterial wall abnormalities, during inflammation and in atherosclerosis (see Abstract, discussion).

The references do not teach that the agent in the treatment of vascular indication increases the production of TGF- β mRNA levels.

Purchio et al. teach that tamoxifen treated PC-3 cells contain more TGF-β mRNA.

It would have been obvious to one of ordinary skill in the art at the time of the invention to treat a vascular indication by the administration of an agent such as tamoxifen that elevates TGF- β levels. The motivation to do is provided by Grainger and Bjorkerud. Grainger teaches that tamoxifen decreased the rate of proliferation of rat vascular smooth muscle cells and induces TGF- β levels in culture and Bjorkerud teaches the role of TGF- β in myodifferentiation and inhibition of growth of smooth

muscle cells in vitro and further teaches that the transforming growth factor could play an important role in the arterial wall abnormalities, during inflammation and in atherosclerosis. Hence one of ordinary skill in the art would have been motivated to select an agent like tamoxifen in a method of treating vascular indication in a mammal to inhibit smooth muscle cell proliferation. The references do not specifically teach determining an agent for TGF-beta elevation or selecting a cytostatic dose of the agent as claimed by applicant. The dosage or selection of an agent or mode of administration is clearly a result effective parameter that a person of ordinary skill in the art would routinely optimize. Optimization of parameters is a routine practice that would be obvious for a person of ordinary skill in the art to employ. It would have been customary for an artisan of ordinary skill to determine the optimal amount of ingredient to add in order to best achieve the desired results. The references do not teach that the smooth muscle cell proliferation is associated with procedural vascular trauma. It would have been obvious to one of ordinary skill in the art to administer an agent in a vascular indication that is associated with procedural vascular trauma. One of ordinary skill in the art would have been motivated to administer an agent that has been shown by Grainger that decreases the proliferation of smooth muscle cells in general to any condition related to smooth muscle cell proliferation in order to achieve similar therapeutic benefits.

Claims 153-155, 169, are rejected under 35 U.S.C. 103(a) as being unpatentable over Fischer et al. (Experimental and Molecular Pathology, 43, 288-296, 1985) in view

of Grainger et al (Biochem J, 1993, 294, 109-112) and further in view of Purchio et al. (U.S. 5,221,620).

Fischer et al. teach connective tissue is a component of the developing plaque that plays a role in the development of atherosclerosis. The reference teaches that synthesis of the total amount of collagen and elastin, components of connective tissue decreased after tamoxifen treatment (Table II).

The reference does not teach that tamoxifen elevates TGF- β levels.

Grainger et al. teach the induction of Transforming Growth Factor β levels and the decrease in the rate of proliferation of rat vascular smooth muscle cells in culture by tamoxifen (see Abstract).

The references do not teach that the agent in the treatment of vascular indication increases the production of TGF- β mRNA levels.

Purchio et al. teach that tamoxifen treated PC-3 cells contain more TGF-β mRNA.

It would have been obvious to one of ordinary skill in the art at the time of the invention to treat a vascular indication by the administration of an agent such as tamoxifen that elevates TGF- β levels. One of ordinary skill in the art would have been motivated to administer an agent such as tamoxifen in a therapeutic method of treating a vascular indication because Fischer teaches the decreased synthesis of connective tissue components which plays a role in plaques in atherosclerosis and Grainger teaches that tamoxifen increases TGF- β levels and decreases the rate of proliferation of rat vascular smooth muscle cells. The references do not explicitly teach determining

an agent for TGF-beta elevation or selecting a cytostatic dose of the agent as claimed by applicant. The dosage or selection of an agent or mode of administration is clearly a result effective parameter that a person of ordinary skill in the art would routinely optimize. Optimization of parameters is a routine practice that would be obvious for a person of ordinary skill in the art to employ. It would have been customary for an artisan of ordinary skill to determine the optimal amount of ingredient to add in order to best achieve the desired results. The references do not teach that the smooth muscle cell proliferation is associated with procedural vascular trauma. It would have been obvious to one of ordinary skill in the art to administer an agent in a vascular indication that is associated with procedural vascular trauma. One of ordinary skill in the art would have been motivated to administer an agent that has been shown by Grainger that decreases the proliferation of smooth muscle cells in general to any condition related to smooth muscle cell proliferation in order to achieve similar therapeutic benefits.

Claims 153-159, 164, 165, 168 are rejected under 35 U.S.C. 103(a)as being unpatentable over Sawada et al. (Pharmacometrics, 1992) in view of Ellis et al. (U.S. 4,826,876).

Sawada et al. teach the administration of toremifene citrate (NK622) in 0.1 mg/kg or more including 10mg/kg (cytostatic dose) to female rats showed decrease in total cholesterol in rats.

Sawada et al. do not teach a method of treatment of a mammal with cardiovascular or vascular indication (atherosclerosis) or mechanism of increasing the

level of TGF-beta to decrease lesion formation or inhibition of lipid accumulation, and dosage formulation set forth in claim 153.

Ellis et al. teach that patients having elevated plasma lipid levels are considered at risk of developing coronary heart disease or other manifestations of atherosclerosis as a result of their high plasma cholesterol concentrations. The reference further teaches that anti-hyperlipidaemic agents, which lower the ratio of LDL-cholesterol to HDL cholesterol, are indicated as anti-atherosclerotic agents (col. 17, lines 51-57).

It would have been obvious to one of ordinary skill in the art to employ toremifene citrate (NK622) in 0.1 mg/kg or more including 10mg/kg (cytostatic dose) to a mammal at risk or afflicted with cardiovascular or vascular indication such as atherosclerosis. One would have been motivated to employ toremifene citrate (NK622) in 0.1 mg/kg or more including 10mg/kg (cytostatic dose) to a mammal at risk or afflicted with cardiovascular or vascular indication such as atherosclerosis because Sawada et al. teach the administration of toremifene citrate (NK622) in 0.1 mg/kg or more including 10mg/kg (cytostatic dose) to female rats showed decrease in total cholesterol in rats and Ellis teach that elevated lipid levels are considered at risk of developing coronary heart disease. One would be further motivated to make such a modification in order to achieve an expected benefit of lowering total cholesterol level in a mammal suffering from atherosclerosis. The references do not specifically teach determining an agent for TGF-beta elevation or selecting a cytostatic dose of the agent as claimed by applicant. That applicant may have determined a mechanism by which the active ingredient gives increasing the level of TGF-beta to decrease lesion formation or inhibition of lipid

accumulation does not alter the fact that the compound has been previously used to obtain the same pharmacological effects (lowering total cholesterol) which would result from the claimed method upon the administration of same active agent in a same amount to the mammal in need thereof. An explanation of why that effect occurs does not make novel or even unobvious the treatment of the conditions encompassed by the claims. The pharmaceutical forms, e.g., sustained release, immediate release etc; mode of administration, flavors, surfactant are all deemed obvious since they are all within the knowledge of the skilled pharmacologist and represent conventional formulations and modes of administration.

Claims 153-155, 157-159, 164, 165, 168 are rejected under 35 U.S.C. 103(a)as being unpatentable over Gylling et al. (Atherosclerosis, 96, 1992, 245-247) in view of Ellis et al. (U.S. 4,826,876).

Gylling et al. teach that tamoxifen decreases serum cholesterol by inhibiting cholesterol synthesis (Fig 1). The reference further teaches that a long-term use of tamoxifen as adjuvant therapy has revealed a cardio protective estrogen-like effect (p 245, col.1, lines 4-7).

The reference does not teach a method of treatment of a mammal with cardiovascular or vascular indication (atherosclerosis) or mechanism of increasing the level of TGF-beta to decrease lesion formation or inhibition of lipid accumulation, and dosage formulation set forth in claim 153.

Ellis et al. teach that patients having elevated plasma lipid levels are considered at risk of developing coronary heart disease or other manifestations of atherosclerosis

as a result of their high plasma cholesterol concentrations. The reference further teaches that anti-hyperlipidaemic agents, which lower the ratio of LDL-cholesterol to HDL cholesterol, are indicated as anti-atherosclerotic agents (col. 17, lines 51-57).

It would have been obvious to one of ordinary skill in the art to employ tamoxifen to a mammal with cardiovascular or vascular indication such as atherosclerosis. One would have been motivated to employ tamoxifen to a mammal at risk or afflicted with cardiovascular or vascular indication such as atherosclerosis because Gylling teaches the decrease in serum cholesterol in a patient with breast cancer. The reference further teaches that a long-term use of tamoxifen as adjuvant therapy has revealed a cardio protective estrogen-like effect. Ellis teaches that elevated lipid levels are considered at risk of developing coronary heart disease. One would be further motivated to make such a modification in order to achieve an expected benefit of lowering total cholesterol level in a mammal suffering from atherosclerosis. The references do not specifically teach determining an agent for TGF-beta elevation or selecting a cytostatic dose of the agent as claimed by applicant. That applicant may have determined a mechanism by which the active ingredient gives increasing the level of TGF-beta to decrease lesion formation or inhibition of lipid accumulation does not alter the fact that the compound has been previously used to obtain the same pharmacological effects (lowering total cholesterol) which would result from the claimed method upon the administration of same active agent in a same amount to the mammal in need thereof. An explanation of why that effect occurs does not make novel or even unobvious the treatment of the conditions encompassed by the claims. The pharmaceutical forms, e.g., sustained release,

immediate release etc; mode of administration, flavors, surfactant are all deemed obvious since they are all within the knowledge of the skilled pharmacologist and represent conventional formulations and modes of administration.

Conclusion

No Claims are allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Umamaheswari Ramachandran whose telephone number is 571-272-9926. The examiner can normally be reached on M-F 8:30 AM - 5:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Sreeni Padmanabhan can be reached on 571-272-0629. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

SREENI PADMANABHAN
SUPERVISORY PATENT EXAMINER